HOUSEHOLD RISKS WITH INORGANIC FIBERS*

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In reviewing the question of mineral fibers, it is well to start historically. The first case of asbestosis was seen in 1898 by Dr. Montague Murray of Charing Cross Hospital in London. He saw a man who was short of breath, and who died. He told Dr. Murray that he had worked in one of the new asbestos factories in London; the industry had started around 1880. When he died, the autopsy showed diffuse interstitial fibrosis. At that time we used very little asbestos in our country, approximately 6,000 tons a year. We now use approximately 600,000 tons a year.

Following that case, we observed the experience of the growing asbestos industry and accumulated knowledge. One of the things we learned was that, unlike the situation with other dusts, the pleura was often involved when asbestos was inhaled. This does not happen with silica, with diatomaceous earth, with aluminum, coal, carbon black. For reasons that we still do not know, with asbestos the pleura will often show fibrotic plaques — sometimes calcified. They are an indication of asbestos inhalation. They need not produce any disability unless they are thick or completely encircle the lung.

In 1935, at about the time we began to learn about the pleura, Kenneth Lynch, then professor of pathology at the Medical University of South Carolina, reported a very unusual case, a man who had carcinoma of the lung and also asbestosis.

The editor of the *American Journal of Cancer* thought this worth publishing.[1] In 1935 — one may not now appreciate it — lung cancer was a rare disease. There was good reason: not many people were smoking

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cigarettes in 1900 or 1910. Also, since there was not much asbestos used in 1910, there were not many people in 1935 who died of asbestosis. Dr. Lynch posed the possibility of an association between the asbestosis and the lung cancer.

Not long after that, in the mid-1940s and 1950s, other associations began to be seen, when malignancies of the pleura were found in people who had been exposed to asbestos. The pleura, of course, is the mesothelial lining of the chest, so its tumors are called mesothelioma. It was a very rare disease before World War II.

When I first came to Mount Sinai at the beginning of the 1940s, a debate was going on. In 1931 the first modern paper on the pathology of mesothelioma had been written by Dr. Paul Klemperer and Dr. Coleman Rabin. They reported two cases at Mount Sinai and three more from the German literature. Dr. Sadao Otani, our surgical pathologist, disagreed. Two cases? He felt it probably a variant of another neoplasm. For the next years this discussion continued. It seemed an empty debate because few such cases were seen. We see them now, unfortunately. Dr. Otani was wrong, because I see almost a case a week of diffuse malignant pleural or peritoneal mesothelioma — invariably fatal, generally within a year, rarely two, three, four, five years.

The pace increased during the 1950s, when more and more of these neoplasms began to be seen and questions had to be answered. They were using epidemiological techniques learned in the cohort smoking studies of the American Cancer Society.

What was done was to investigate a group of people known to be exposed to asbestos. In 1963 there was a small union of insulation workers in the New York metropolitan area, with 1,250 members. More than 1,100 were examined, primarily for signs of asbestosis. About half had abnormal films. But what was interesting was that, of the 725 with less than 20 years from onset of exposure, most had normal roentgenograms. It was only after the 20-year point that most roentgenograms became abnormal, not infrequently extensively so. These results have been considered the "20-year rule" (Table I). This was also found to be true for the pleura. In the first 20 years from onset of exposure, most workers had normal roentgenograms insofar as the pleura was concerned. After the 20-year point, fibrosis or calcification or both became common (Table II).

To see whether Dr. Lynch was correct or incorrect, a cohort study was

TABLE I

X-RAY CHANGES IN ASBESTOS INSULATION WORKERS

Onset of exposure (yrs.)	*No.*	*% Normal*	*% Abnormal*	*Asbestosis (grade) 1*	*2*	*3*
40+	121	5.8	94.2	35	51	28
30-39	194	12.9	87.1	102	49	18
20-29	77	27.2	72.8	35	17	4
10-19	379	55.9	44.1	158	9	0
0-9	346	89.6	10.4	36	0	0
	1,117	51.5	48.5	366	126	50

TABLE II

ROENTGENOGRAPHIC EVIDENCE OF PLEURAL ABNORMALITY AMONG 1,117 ASBESTOS INSULATION WORKERS

Years from onset of exposure	*Number examined*	*Normal pleura*	*Asbestosis (grade) Fibrosis*	*Calcification*
40+	121	28	65	70
30-39	194	96	62	67
20-29	77	47	25	8
10-19	379	340	36	5
0-9	346	342	4	0

done. A list was made of the 632 men in this union on January 1, 1943. Each has been traced and each is still under observation.[2,3] By 1977 there should have been 329 deaths; instead, there were 478. Rather than 57 deaths of cancer, there were 210; and, instead of 13 deaths of lung cancer, there were 93. One of every five of these men died of lung cancer (Table III). Instead of no deaths of mesothelioma (in general, this has been so rare in the past that it has not been separately coded in the International Classification of Causes of Death), there were 38. They were excess deaths from gastrointestinal cancer, as well as deaths from asbestosis.

This was a small study. Therefore, on January 1, 1967 the entire membership of this union in the United States and Canada was registered. There were 17,800 men on its rolls on that day. By 1977, after some

TABLE III

EXPECTED AND OBSERVED DEATHS AMONG 632 NEW YORK-NEW JERSEY ASBESTOS INSULATION WORKERS JANUARY 1, 1943-DECEMBER 31, 1976
(13,925 Man-years of Observation)

Underlying cause of death	*Expected**	*Observed*
Total deaths, all causes	328.9	478
Total cancer, all sites	57.0	210
Cancer of lung	13.3	93
Pleural mesothelioma	†	11
Peritoneal mesothelioma	†	27
Cancer of esophagus	1.4	1
Cancer of stomach	5.4	19
Cancer of colon-rectum	8.3	23
Cancer of larynx, pharynx, buccal cavity	2.8	6
Cancer of kidney	1.3	2
All other cancer	24.5	28
Noninfectious pulmonary diseases, total	9.3	45
Asbestosis	†	41
All other causes	262.6	223

*Expected deaths are based upon white male age-specific U.S. death rates of the U.S. National Center for Health Statistics, 1949-1976. Rates for specific cause of death for 1943-1948 were extrapolated from rates for 1949-1955.

†Rates are not available, but these have been rare causes of death in the general population.

166,000 man-years of observation, there should have been 1,659 deaths. There were 2,271. Instead of 320 deaths from cancer, there were 995. Instead of 106 deaths from cancer of the lung, there were 486, again one of every five. And, instead of no deaths from mesothelioma, there were 175.[3] Gastrointestinal cancer deaths (esophagus, stomach, colon, rectum), cancer of larynx, oropharynx, kidney were increased, as shown in Table IV.

So Dr. Lynch had been right. His hunch at the autopsy table had been correct.

PRODUCT USE

This experience emphasized something else. Until then, studies had primarily been directed at "asbestos workers." But the problem is not only among asbestos workers. For every man or woman who makes an asbestos product — a miner or miller or factory worker — there are at least 100 who are exposed to the dust in use of the products. The major problem was associated with their use.

TABLE IV

DEATHS AMONG 17,800 ASBESTOS INSULATION WORKERS IN THE UNITED STATES AND CANADA JANUARY 1, 1967-DECEMBER 31, 1976

Underlying cause of death	*Expected**	*Observed*		*Ratio o/e*	
		(BE)	*(DC)*	*(BE)*	*(DC)*
Total deaths, all causes	1,658.9	2,271	2,271	1.37	1.37
Total cancer, all sites	319.7	995	922	3.11	2.88
Cancer of lung	105.6	486	429	4.60	4.06
Pleural mesothelioma	**	63	25	...	...
Peritoneal mesothelioma	**	112	24	...	...
Mesothelioma, n.o.s.	**	0	55	...	...
Cancer of esophagus	7.1	18	18	2.53	2.53
Cancer of stomach	14.2	22	18	1.54	1.26
Cancer of colon-rectum	38.1	59	58	1.55	1.52
Cancer of larynx	4.7	11	9	2.34	1.91
Cancer of pharynx, buccal	10.1	21	16	2.08	1.59
Cancer of kidney	8.1	19	18	2.36	2.23
All other cancer	131.8	184	252	1.40	1.91
Noninfectious pulmonary disease, total	59.0	212	188	3.59	3.19
Asbestosis	**	168	78	...	...
All other causes	1,280.2	1,064	1,161	0.83	0.91

Number of men	17,800
Man-years of observation	166,853

* Expected deaths are based upon white male age-specific U.S. dath rates of the U.S. National Center for Health Statistics, 1967-1976.

** Rates are not available, but these have been rare causes of death in the general population.

BE—Best evidence. Number of deaths categorized after review of best available information (autopsy, surgical, clinical).

DC—Number of deaths as recorded from death certificate information only.

CLINICAL LATENCY

In the larger study it became possible to see whether the 20-year rule held for cancer. It did. Analyzed by the number of years subsequent to onset of exposure, the ratio between expected and observed lung cancer rates showed no great difference until about 15 years had passed, going up sharply at around 30, 35 years. These people begin work at 17, 18, 20. They don't die until they are 50 or 55, like most people with cancer (Table V).

For mesothelioma, too, one sees very little before 20 years from onset. The death rates per 1,000 person-years at risk rises markedly at around 30, 35 years. At age 55, for example, 35 years from onset, one out of every three deaths is due to lung cancer, and roughly one out of 10 is due to mesothelioma. The problem of clinical latency, with us in almost everything we are discussing these days, is of major significance.

PLEURAL ABNORMALITIES

The prevalence of pleural disease is important for the internist, for the diagnostician, for the hospital physician. Calcifications of the diaphragm are pathognomonic diagnostic stigmata. Pleural plaques and pleural fibrosis may be the only evidence of previous asbestos exposure. There need be no simultaneous parenchymal fibrosis. Such cases generally have no symptoms.

Latency can perhaps be even better illustrated clinically than statistically. I saw a man in 1951 with a fairly normal chest roentgenogram. He was slightly short of breath. There was no obvious cause. When he came to see me five years later, in 1956, he had clear evidence of bilateral lower lobe interstitial fibrosis. I said, "Bob, you have changes that look like you might have been exposed to asbestos." He had told me he had been a machinist and a truck driver, but had not worked with asbestos. His wife spoke up at that point. She said, "Bob, don't you remember when we first got married, in 1935, you worked at the Worldbestos Corporation, weaving brake linings?" He answered, "Oh, that. That was only for six months." His film in 1979 showed extensive changes. He cannot breathe very well. He has moved to Florida; sits on the porch and watches the world go by. He had worked for six months with asbestos. The dust which had entered his lungs in 1935 had been there 25 years in 1956.

TABLE V

DEATHS AMONG 17,800 ASBESTOS INSULATION WORKERS IN UNITED STATES AND CANADA, JANUARY 1, 1967-DECEMBER 31, 1976.
ANALYSIS BY DURATION FROM ONSET OF EMPLOYMENT

Duration from onset (Years)	Number of men	Person-years of observation	Lung cancer					Pleural mesothelioma			Peritoneal mesothelioma		
			Exp.*	Observed (BE)	Observed (DC)	Ratio o/e (BE)	Ratio o/e (DC)	Number (BE)	Number (DC)	No./1,000 person-years (BE)	Number (BE)	Number (DC)	No./1,000 person-years (BE)
10	8,190	26,393	0.7	0	0	—	—	0	0	0	0	0	0
10—14	9,063	29,003	2.7	7	5	2.55	182	0	0	0	0	0	0
15—19	9,948	34,066	8.5	29	27	3.40	3.17	2	2	0.06	3	0	0.09
20—24	8,887	31,268	17.0	59	57	3.48	3.36	6	4	0.19	3	2	0.10
25—29	6,596	20,657	21.0	105	96	5.00	4.58	13	5	0.63	19	3	0.92
30—34	3,547	11,598	18.4	112	103	6.08	5.59	9	3	0.78	23	6	1.98
35—39	2,020	5,403	11.5	65	57	5.68	4.98	15	4	2.78	19	5	3.52
40—44	1,108	3,160	8.1	40	131	4.93	3.82	4	3	1.27	16	3	5.06
45+	1,448	5,305	17.8	69	53	3.89	2.98	14	4	2.64	29	5	5.47

*Expected deaths are based upon white male age-specific U.S. death rates of the U.S. National Center for Health Statistics, 1967-1976: Smoking habits not taken into account.

BE—Best evidence. Number of deaths categorized after review of best available information (autopsy, surgical, clinical).

DC—Number of deaths as recorded from death certificate information only.

INTERACTION WITH SMOKING

Not everybody who has been significantly exposed to asbestos dies of lung cancer. Most do not; if one out of five does, four out of five do not. Some people develop mesothelioma and some do not. We have information now why some get lung cancer and others do not.

We decided to study the relationship between cigarette smoking and asbestos exposure. Like many blue-collar workers, asbestos-exposed workers also frequently smoke cigarettes. When we did our survey in 1963, each man was asked about his smoking habits. Three hundred and seventy men were at least 20 years from onset of exposure by 1963 (many 30 to 40 years) and thus very much at risk of dying of lung cancer. There were 87 men who had never smoked cigarettes and 283 who had. We did not expect many lung cancers among the nonsmokers. By 1967 there were none. When they died of other things and their lungs were examined, there was much asbestos. They did not, however, die of lung cancer. On the other hand, among the 283 smokers we expected three deaths of lung cancer. Instead, there were 24. In other words, it was not the asbestos alone. None of the nonsmoking asbestos workers died of lung cancer. It was not the cigarette smoking alone. Only three would have died of lung cancer. The combination of the two, the multiple-factor interaction, gave the extraordinary increase.[4]

We did a similar study in the group of 17,800 men. When they were registered, their smoking habits were also recorded. For our controls, we studied more than 73,000 men in the American Cancer Society Cancer Prevention Study[5] who were very much like the asbestos workers, i.e., they were white, they were not farmers, and, on registration in 1960, they had recorded that they were exposed at their work to dust, fumes, vapors, radiation, or chemicals. They had no more than a high school education, were alive on January 1, 1967, and we knew their smoking habits. By 1977 we found that of the people who did not work with asbestos and did not smoke cigarettes, the death rate for lung cancer was 11 per 100,000 per year, standardized for age and smoking habits. For the asbestos workers who did not smoke, the death rate was 58 per 100,000 per year, five times as much. On the other hand, for those who smoked but did not work with asbestos, it was 122 per 100,000 per year, and for those who had both exposures, the rate was 601 per 100,000 per year — evidence of the extraordinary importance of multiple factor interaction (Table VI).[6]

TABLE VI

AGE-STANDARDIZED LUNG CANCER DEATH RATES* FOR CIGARETTE SMOKING AND/OR OCCUPATIONAL EXPOSURE TO ASBESTOS DUST COMPARED WITH NO SMOKING AND NO OCCUPATIONAL EXPOSURE TO ASBESTOS DUST

Group	*Exposure to asbestos?*	*History cigarette smoking?*	*Death rate*	*Mortality difference*	*Mortality ratio*
Control	No	No	11.3	0.0	1.00
Asbestos workers	Yes	No	58.4	+47.1	5.17
Control	No	Yes	122.6	+111.3	10.85
Asbestos workers	Yes	Yes	601.6	+590.3	53.24

*Rate per 100,000 man-years standardized for age on the distribution of the man-years of all the asbestos workers. Number of lung cancer deaths based on death certificate information.

MESOTHELIOMA AND ASBESTOS

After the report of the two cases of mesothelioma in 1931 and the differences of opinion, mesothelioma was sought at Mount Sinai. There were three more cases during the next 30 years, evidence that it was a rare disease. It was at this point we had our first bad surprise. In 1960 J.C. Wagner, a pathologist at the Pneumoconiosis Research Unit in Johannesburg, South Africa, reported that he saw 47 cases of mesothelioma![7] Most patients were dead. They had all been seen in one part of South Africa, the northwestern portion of the Cape Province, a part of South Africa with many small crocidolite asbestos workings. Dr. Wagner visited the relatives of these people and found that in 45 of the 47 there had been, often 30 to 35 years before, opportunity for asbestos contact, although not necessarily while working the material. Some exposures were relatively minor, "environmental" in nature. This suggested the possibility for the first time that one did not need much asbestos to cause cancer — only enough to start it — it then had a life of its own.

With this knowledge, Muriel L. Newhouse, an epidemiologist at the London School of Hygiene, examined the 76 cases in the files of the London Hospital. She also visited relatives. She found that 31 of the 76 had worked with asbestos. That came as no surprise; by 1965 we knew that people with mesothelioma had often worked with asbestos. But of the 45 who had not worked with asbestos, nine had lived in the household of an asbestos worker. These were often women who had washed their hus-

bands' clothes when they came home from work. Eleven had merely lived within a half mile of one of the asbestos plants in London, at Barking.[8] This again suggested that one did not necessarily need much asbestos to produce mesothelioma.

Family Contact Asbestos Disease

This, then, addressed a subject we are now discussing, the problem of family contact disease.

We have been tracing the wives and children of workers employed in an asbestos plant in Paterson, N.J., that operated from 1941 to 1954. The workers have been dying of the usual asbestos diseases.[9] In tracing workers we contacted the wives and children. We have, so far, examined approximately 750. In the first 626, one third had abnormal roentgenograms: pleural plaques, pleural fibrosis, sometimes even interstitial fibrosis of limited extent.[10]

B.G. was an example of family contact disease. She was at our hospital in 1978. She had had a normal chest roentgenogram in 1974. It was taken because her mother had died of mesothelioma. Her father had died of lung cancer; he had been a shipyard worker in Massachusetts. A roentgenogram taken in 1978 showed mesothelioma, from which she died. When she was seen she told us that when her father came home from the shipyard, her mother took his clothes and shook them, with the children playing on the floor nearby.

In the factory population that we are tracing, among the 933 workers, of the first 304 deaths, more than 20 years from onset of employment, we have seen 14 mesotheliomas — approximately 5%. In the first 384 deaths studied among the family contacts, more than 20 years from onset, four were due to mesothelioma — approximately 1%. Equally worrisome, it appears that there will be a significant increase in lung cancer rates in the same group. William J. Nicholson and his colleagues calculate that about nine million workers were significantly exposed from 1940 to 1980 and are currently alive.[11] There probably are a similar number of wives and children.

There have been other sources of asbestos exposure in the home — among the do-it-yourselfers, for example, the use of spackle compound to tape joints on wall board. No one had told them, over the years, that most spackle compounds contained 12 to 15% asbestos.[12] Some papier maché used by the New York City Board of Education until recently con-

tained approximately 50% asbestos. When we went to the New York City Board of Education with this information, it was found that they had purchased 50,000 five-pound bags of such papier maché material for kindergarten use. These have been discarded.

There have been asbestos textiles in the home, such as asbestos gloves. There have been do-it-yourself brake repair work and the do-it-yourself carpenter with asbestos cement board. Many furnaces and their pipes have been insulated with asbestos, needing repair and maintenance — sometimes do-it-yourself.

One of the most curious situations we have encountered was a young woman who called me saying, "My name is so and so. I live in Connecticut. I have just bought a coat and the label says it has 7% asbestos." I said, "Quit your kidding; people are seeing asbestos under every bed." She said, "No, I was in Boston and went to a store with a famous basement, and it was a very good buy." She was right; it was a good buy and it did contain 7% asbestos. When we sampled air near the coat, levels were higher than in some factories. When we washed the coat with other clothes in the same tumbling machine, they became contaminated with asbestos.

We then discussed this with the International Ladies Garment Workers Union. Their members were cutting the fabric. They identified the importer. He was very clever. He knew that when a fabric that has a new fiber was imported, the rate of duty reflected the new fiber. Asbestos fiber had a much lower level of import duty. I have been told that a million dollars was saved by putting in 7% asbestos.

As a postscript, one might mention that household contact mesothelioma with another mineral fiber—erionite (a fibrous zeolite)—is now being seen in Cappadocia, Turkey, where the mineral is used for household construction.[13]

Questions and Answers

MR. HARVEY SACHS: (Princeton University) Erionite is a common accessory mineral in the area of Nevada proposed for the MX missile sites, which will require enormous amounts of excavation.

DR. SELIKOFF: You are correct. Erionite is found in many parts of the United States. It has not been used very much commercially until recently. Zeolites in general—many of them synthetic—are the molecular sieves of chemistry. There are other fibrous zeolites—clinoptinolite and

others—which can have a fibrous habit. If the MX missile sites are built, we might have to work very large areas of Nevada and Utah, some of which have erionite mineral beds. This is a problem that has been little discussed with regard to placing the MX missiles.

DR. JAMES REPACE (United States Environmental Protection Agency): A number of factors seem to indicate potentially serious public health problems: the large interactive and multiplicative factors for coexposure of tobacco smoke and asbestos; the very extensive use of asbestos around ventilating systems in buildings (I have seen entire floors surfaced with asbestos to keep the heat tansmission down); the fact is that most ventilating systems are made from sheet metal and overlap and there is a lot of leakage and negative pressure so that the shaking from the fan motors can cause induction of the asbestos fibers into the ventilating system and rapid distribution throughout the building.

DR. SELIKOFF: This is potentially an important problem. We do not know yet how to handle it. Not only does negative pressure suck dust into the air circulating system, but many new buildings have the return air plenum spaces between floors. These spaces may be lined with sprayed asbestos. When repairs or maintenance are needed, electricians, sheet metal workers, steam fitters, and others must work in these spaces. Asbestos can be loosened and can contaminate the area.

The problem of maintenance and repair within these buildings is critically important. I am not sure how it will be managed properly. We do not have the engineering technology at the moment to do this readily.

DR. DEMETRIOS MOSCHANDREAS (GEOMET Technologies, Inc.): Would you address yourself to the control methodologies used to eliminate asbestos in schools? The Environmental Protection Agency is now undertaking a rather large program. Should the asbestos be totally removed or are the sealants sufficient to reduce or eliminate the problem?

DR. SELIKOFF: I once knew a fellow student who was not very bright but nevertheless passed all of his exams. When asked a difficult question, he thought and thought, and said, "It all depends."

With asbestos in schools, it all depends. Sometimes, as when it is very firm and in good condition and one can't get to it very easily, it can simply be covered and left undisturbed. A false ceiling may be placed underneath. When repairs have to be made, or the school is going to be remodeled, precautions would then still have to be taken. If, on the other hand, it is friable, it would have to be managed otherwise, often by removal.[14]

We are very fortunate in New York City in having, both in our Environmental Protection Administration and in our Board of Education, very capable people who have been attacking this problem with vigor. The group assigned by the mayor to look at the asbestos problems in New York City schools has been doing a very good job. However, it is going to cost the city many millions of dollars.

SPEAKER: What has the experience been in epidemiology of glass fiber workers?

DR. SELIKOFF: So far, we know little of the cancer potential in humans. Fine glass fibers, i.e., fine enough to enter the recesses of the lung and perhaps be brought to the pleura, have been available for too short a time to give useful information. Experimentally, one can produce mesothelioma with glass fibers, but we do not have adequate human experience yet to evaluate the problem epidemiologically.

DR. BERNERD BURBANK (McGraw-Hill): Do you know of any cases of mesothelioma that are not definitely associated with asbestos exposure?

DR. SELIKOFF: Yes. I am aware of cases in which we cannot identify asbestos exposure. However, we recognize that it is very difficult to obtain accurate histories of what people did 30, 35, 40 years ago. It is difficult to be confident that the second wife or a third cousin really knows what a patient did for six weeks, 35 years ago.

There is an excellent study that bears on this matter, by Cochrane and Webster at the Johannesburg General Hospital in South Africa. When a positive biopsy was obtained for mesothelioma, they visited the patient. Speaking to the patient, not to the next of kin, in 70 consecutive cases over several years they found potential asbestos exposure in 69 of the 70.[15]

There likely are other potential causes. I cannot concede that the pleura will react only to one agent. I am concerned that there may be other causes not recognized at present that may become more and more important. We do not know or suspect what they are. I hope fibrous glass will not turn out to be one of them.

MR. STEPHEN WILDER (Sierra Club): You mentioned the multiplicative effect of cigarette smoking in asbestos exposure. Would it be fair to say that anybody who thinks that he has been exposed to asbestos ought to stop smoking?

DR. SELIKOFF. Without question. You are correct. Our studies show that when asbestos workers stopped smoking, within five to 10 years their risk of dying of lung cancer went to one third to one half of that of their

colleagues who continued to smoke.[16]

This raises ethical and legal questions. Now that we know that people who have been exposed to asbestos should stop smoking, do we have the ethical obligation to identify and to locate these people to advise them about this risk, giving them the chance to stop smoking, to decrease their risk of dying of lung cancer? Is this a legal responsibility as well?

MR. WILDER: May I follow that up? Hasn't everyone who is living and working in midtown Manhattan, where they used to spray asbestos, been exposed?

DR. SELIKOFF: Probably. None of us should smoke cigarettes.

SPEAKER: There is a product that is used in millions of water softeners called sodium zeolite. Is that zeolite of the same type?

DR. SELIKOFF: No. There are many synthetic zeolites. Zeolites are remarkable chemicals, probably some of the most important now being used in industry. We do not know that nonfibrous, granular zeolites will cause the same kinds of lesions as fibrous zeolites such as erionite, which is similar to asbestos in size and shape. We do not know that other zeolites, now widely used in the petrochemical industry, will have the same effect.

You have raised a very important question. I have recommended that research rapidly be done to see whether the zeolites being used now will be hazardous.

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